

# Analysis of Antibiotic Resistivity Pattern of *ESCHERICHIA COLI* Isolated from Clinical Samples in Auchi Township Area, Edo State, NIGERIA

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## ABSTRACT

Resistance to available antimicrobial agents has become a popular finding and spread continues to widen. Despite this fact, studies investigating a common bacterial like *Escherichia coli* (*E. coli*) is lacking in growing rural settings. In this regards, different clinical samples from different health care centers in Auchi Township in Etsako West Local Government Area, Edo State, Nigeria, were screened for *E. coli* and the antibiotic resistivity pattern investigated. A cross section study design was conducted in a bid to achieve this objective. Following standard procedures different human samples were collected and *E. coli* isolated. Antibiotic susceptibility testing was carried out on the isolated *E. coli* using standard microbiology techniques. The data was then subjected to simple descriptive statistics of frequencies and percentages and presented in tables and charts. The results showed that 33.03% of the clinical samples were positive to *E. coli* organism which was most prevalence in stool sample (60.0%). The antibiogram typing of the isolated *E. coli* showed that no two isolate was the same. It was observed that *E. coli* isolates were highly resistance to many of the available antibiotics except for Gentamicin (55.56%), Nitrofurantoin (55.56%), Ofloxacin (52.78%), Ciprofloxacin (55.56%), Perflacin (61.11%) and Cefotaxime (69.44%) where susceptibility exceeded 50%. In addition, *E. coli* isolates from different clinical samples were highly multidrug resistant. These findings suggest phenotypic modifications of *E. coli* bacteria and this may accounts for the resistance potentials and reemerging multidrug resistance. Antibiotics surveillance within and between geographical vicinities is therefore recommended, whose significance cannot be overemphasized.

**Key words:** *Escherichia coli*, Antibiogram typing, Antibiotic, Sensitivity, Resistivity.

## INTRODUCTION

Man and animals in their life times are sure to be infected by one pathogen or the other at any particular time and as such the fight against these pathogens are real and continuous [1]. While antimicrobial agents have made definite impact in the management of these pathogenic diseases, the emergence of resistance strains has threaten available antibiotics on the other hand. The emergence and re-emergence of the multidrug resistant (MDR) microorganism has become an impediment to antibiotic treatment and resulted to the search and need for new antibiotics [2]. According to several studies, the emergence and reemergence of multiple resistance microorganisms in both community and hospital-acquired infections worldwide are threat to antibiotics treatment of infectious diseases and buttress the need for active and continuous surveillance, specific laboratory diagnosis before antibiotic prescription [3-6]. In this regards, resistance of pathogenic organisms to countenance antibiotics has become a worldwide problem with serious consequences on the treatment [7].

One very important organism with several reported multidrug resistant and of public health significance is the *Escherichia coli* (*E. coli*). *E. coli* has widely been implicated in various clinical infections as hospital acquired and community infections [8]. It is one of the organisms most frequently isolated from different clinical cases [9, 10] and has been globally identified in isolates from environmental, animal and human sources [11]. Community and healthcare acquired multi-resistant species of *Streptococci*, *Pseudomonas* and *E. coli* are reported and are said to be on the increase globally [3, 4, 6]. *E. coli* has been linked to well-known antibiotic-resistant gene pools and these genes are transferred into the normal flora of humans and animals,

where they exert a strong selective pressure for the emergence and spread of resistance in *E. coli* strains [12]. Antibiotics treatment has brought about phenotypic changes in *E. coli* and other organisms, often due to chromosomal mutations and resulted in antibiotic resistance [11].

Pathogenic isolates of *E. coli* are reported to have relatively high potentials for developing resistance [13]. In fact, reports from several parts of the globe showed temporal trends in the prevalence of antibiotic resistance among *E. coli* specifically and other microbes at large. During the last 15 years, studies have showed increasing resistance to several commonly used antibiotics [9, 14]. Worrysome, *E. coli* strains causing UTI are said to be often multidrug resistant- refractive to three or more different classes of antibiotics [15]. To this effect, Bhattacharyya et al. [16] suggested that proper susceptibility data from specified area is needed if empirical antibiotics are to be administered in this regards. On the other hand, antibiogram typing is of clinical significance as it establishes about local susceptibility rates, serves as an aid in selecting empiric antibiotic therapy, and in monitoring resistance trends over time within a geographical location [17]. In the words of Fridkin et al. [18], an antibiogram shows the aggregate number of bacteria tested against antimicrobials and incorporates the extent of bacterial isolates vulnerable to every antimicrobial operator tested. This study therefore aimed at investigating the antibiotic resistivity pattern of *E. coli* isolated from different clinical samples in Auchi township area of Edo State, Nigeria via determining their antibiogram typing. This study is significance as it is the first of its kind in the study area. Moreover, antibiogram typing can provide information on the local resistance pattern,

while also supporting the use of optimal empiric treatment and identifying opportunities to reduce inappropriate antibiotic usage [18-20].

## MATERIALS AND METHODS

**Study area:** This study was carried out in Auchi township (7°04'N, 6°16'E) in Edo State, Nigeria between 15<sup>th</sup> of January to 15<sup>th</sup> of April 2015. The area is the headquarters of the Etsako West Local Government Area and a host of a tertiary institution (Auchi Polytechnic Auchi), several secondary and primary school, several clinic, laboratories and government health care facilities.

**Ethical consideration:** Approval was obtained from the health facilities used. Once a patient was identified as suitable, the study was explained to the patient (in English and native language for those who do not understand English) and consent to be included in the study obtained. The study was explained to the patients and they were told they can decline and decide to be excluded from the study at any point in time without consequence on the services they received from the health facility. The study was conducted in compliance with the Declaration on the Right of the subject/participant as stated in WMA, 2000.

**Sampling:** A total of 109 clinical specimens which includes urine, blood, stools and swab (wound, throat, and ear swabs) were collected from randomly selected clinics consisting of a private clinic (Fate Medical Center), a government owned health facility (General hospital) and a medical laboratory (Medi-view laboratory) in Auchi Township. Specimens were transported to the diagnostic laboratory of Fate Foundation Hospital and stored at 4°C refrigerator before culturing, isolation and provisional identification. Analysis was done within 24hrs of collection. Isolates were placed on Nutrient agar slopes and stored in the refrigerator. Periodically, isolates were transferred to the microbiology laboratory at Ambrose Alli University Ekpoma for confirmatory identification.

**Identification of Isolates:** Isolates were cultured on MacConkey agar and nutrient agar under aseptic conditions [21-23]. The MacConkey agar and Nutrient agar plates were incubated at 37°C. After overnight incubation, growths of suspected organisms were identified by their colonial morphology and biochemical characteristics [24]. These tests include the following;

1. **Gram Stain:** Discrete colonies of the test organisms were obtained aseptically with sterile wire loop and emulsified in drop of normal saline on a clean sterile glass slide. The smear was air dried and then heat fixed by passing through it slightly through a Bunsen burner flame. Then the fixed smear was covered with crystal violet stain on a staining rack for 30 seconds, and then washed off

with distilled water. The smear was then flooded with lugol's iodine for one minute, and then rinsed with distilled water. The smear was decolourized with acetone for 10 seconds and was immediately washed off with distilled water. The smear was counter stained with neutral red for one minute, washed off and then allow to air dry. A drop of immersion oil was placed on the stain portion of the slide and it was viewed under x100 objective lens. All Gram negative organisms were pinkish or pale to dark red in colour, which is as a result of the secondary dye while gram positive organisms were usually purple in colour which was the colour of the primary dye.

2. **Motility Testing:** A molded plasticine ring was set on a clean slide. A loopful of an overnight broth culture of the test organism was taken onto a cover-slip, and the slide was inverted on the cover-slip making sure that the culture drop was within the plasticine ring but without touching the ring. This was inverted and examined under the x10 and x40 objectives.
3. **Catalase Test:** Catalase test was carried out by placing a drop of hydrogen peroxide on a clean grease free glass slide. A colony of the test organism was taken with a sterile platinum wire loop, and emulsified in the hydrogen peroxide on the slide. The production of gas bubbles which indicated positive result was then observed.
4. **Oxidase Test:** A piece of filter paper was placed on a clean Petri-dish and two drops of freshly prepared oxidase reagent was added. Then a colony of the test organism was smeared on the oxidase reagent soaked filter paper. Development of a deep purple colour within 10 seconds indicated positive result.
5. **Urease Test:** This test the ability of certain organisms to produce the enzyme urease which decomposes urea into alkaline, ammonia and carbon (iv) oxide. The test organism was inoculated heavily in bijoux bottle containing 3ml of sterile Christensen's modified urea broth. It was incubated at 37°C for 12 hours. A pink colour in the medium indicated a positive result.
6. **Coagulase Test:** This test is used to differentiate *Staphylococcus aureus* which produces the enzyme coagulase, from *S. epidermidis* and *S. saprophyticus* which do not produce coagulase. Coagulase causes plasma to clot by converting fibrinogen to fibrin. Two types of coagulase are produced by most strains of *S. aureus*. Free coagulase converts fibrinogen to fibrin by activating a coagulase reacting factor present in plasma and this is detected by the appearance of a fibrin clot in the tube test while bound coagulase (clumping factor) converts fibrinogen directly to fibrin without requiring a coagulase –reacting factor. It can be detected by the clumping of bacterial cells in the rapid slide test. A slide coagulase test is run with a control to rule out auto agglutination. Two drops of physiological saline are put onto a slide labeled with test (T) and control

(C), the two saline drops are emulsified with the test organism using a wire loop. A drop of plasma is placed on the inoculated saline drop corresponding to test, and mixed well, then the slide is rocked gently for about ten seconds and observes for clumping. No clumping is an indication of a negative result. If slide coagulase is negative this is followed by a tube test for further confirmation. The tube test uses rabbit plasma that has been inoculated with Staphylococcal colony (i.e. Gram positive cocci which are catalase positive). The tube is incubated at 37°C for 1 hour. If negative, then incubation is continued up to 18 hours. If positive, the plasma will coagulate.

7. Indole Test: This test is used to demonstrate the ability of certain bacteria to decompose the amino acid tryptophan to indole which accumulates in the medium; the organism is inoculated into peptone water and incubated overnight at 37°C. The presence or formation of indole is tested by added a few drops of Kovac's reagent (0.2-0.3). The test tube is well shaken and allowed to stand, the formation of a dark red colouration indicates a positive result and no colour change indicates a negative result.
8. Citrate Test: Slant of medium containing citrate and ammonium salt was prepared in bijoux bottles and stored at 4°C in the refrigerator, with the use of a sterile wire; the slant was streaked with a saline suspension of the test organism and then stabbed into the medium followed by incubation at 35°C for 48 hours. A bright colour indicated positive result.
9. Indole Test: Bijou bottles containing 3ml of sterile peptone water was inoculated with test organism, and then incubated at 37°C for 48 hours. Thereafter, 0.5ml Kovac's reagent was added and shaken gently. Positive result was indicated by the formation of red colour in the surface layer within 10 minutes.

**Antibiotic susceptibility testing:** Prepared antibiotic discs were used as follows; A 1/100 dilution of an overnight nutrient broth culture of each test organism was poured on the surface of nutrient agar plate that was notched round. The excess broth was discarded into a disinfectant jar. The plates were left to stand on the bench top for 30 minutes. After 30 minutes, the antibiotic discs were placed on the surface of the inoculated agar medium. The plates were incubated at 37°C for 24 hours after which the zones of inhibition were observed and recorded. Zones of up to 13 mm diameter or over were recorded as sensitive while those less than 13 mm were regarded as resistance [21].

**Preparation of antibiotic susceptibility discs:** In cases where the commercially prepared antibiotic discs were not available, the discs were personally prepared as follows: Whatman No.1 filter paper discs with diameter of 6 mm were punched out with a paper puncher. One hundred of the discs were counted into bijoux bottles. The bottles and the discs were sterilized in a hot air oven

at 140°C for 2 hrs. The bottle and its contents were allowed to cool to room temperature. The discs were then aseptically inoculated with 1 ml of the pre-prepared antibiotic solution. The bottles' caps were partly screwed down and allowed to dry overnight in a 37°C incubator [21].

**Antibiogram typing:** This was done by dividing the 21 antibiotics into seven different groups of 3 antibiotics each according to their mode of action, similarities and usage [35]; antibiotics were arranged in ascending order of their molecular weight or their generations in each group of 3 antibiotics viz;

Group 1: Ampicillin, Amoxicillin and Cloxacillin; These are bacteria cell wall synthesis inhibitors. They impair cell wall synthesis by preventing cross-binding of the peptidoglycan polymers necessary for cell wall formation and by binding the penicillin-binding proteins (PBPs) (carboxypeptidases, endopeptidases, and transpeptidases) that participate in cell wall synthesis.

Group 2: Flucloxacillin, Amoxycylav and Ampisulbactam; These are broad-spectrum penicillin with a fluorinated side chain in the first and attached clavulanic acid in the other two

Group 3: Gentamicin, Tobramycin and Streptomycin; These are Aminoglycosides- broad spectrum, protein synthesis inhibitors, active against aerobes only.

Group 4: Chloramphenicol, Tetracycline and Cotrimoxazole; Septrin is a folic acid inhibitor while the other two are protein synthesis inhibitors. Here, they represent the most commonly used (abused) antimicrobials in the community.

Group 5: Metronidazole, Nitrofurantoin and Nalidixic acid; Metronidazole is anti-protozoan but is also active against anaerobic and facultatively anaerobic bacteria; Nitrofurantoin is active against Gram-positive and Gram-negative bacteria and is mostly used as urinary antiseptic while antimicrobial activity of early quinolones (the first generation quinolones) such as Nalidixic acid, are excellent against aerobic Gram-negative bacteria.

Group 6: Ofloxacin, Ciproxin and Peflacin; Quinolones act by inhibiting the action of topoisomerases II (DNA gyrase) and topoisomerase IV. For Gram-negative bacteria the prime target of quinolones is the DNA gyrase, whereas in the Gram-positives it is the topoisomerase IV. These three quinolones act by inhibiting the DNA gyrase.

Group 7: Cephalexine, Cefotaxime and Cefuroxime; Cephalexin is an oral penicillin substitute that is active mainly against Gram-

positive organisms while the latter two are broad spectrum and highly active against Gram-positive and Gram- negative organisms. These drugs are highly expensive, and they are usually not abused.

**Ajumali's method of pneumonic coding [26]:** This is an adaptation of Ajumali's method of pneumonic coding [25,26]. Sensitive result was scored as (+) while resistance was recorded as (-). The three antibiotics in each group were given numerical values of 1, 2, and 4. A perfect sensitivity to the three antibiotics will give a summation of  $1+2+4=7$ . On the other hand, complete resistance to the three antibiotics will give a summation of  $0+0+0=0$ . Other values are obtained by adding up these numerical values; in which case an isolate can receive a score of 0-7 in each triplet segment. Combining the seven-triplet segments together then gives a seven-digit numerical value as the antibiogram type.

**Materials/Instrumentation and Techniques:** The equipment used in this study include the following; hot air oven, incubator, Aluminium foil paper, Petri dishes, measuring cylinder, Bijou bottles, Cotton wool, test tubes, sterile hand gloves, Masking tape, glass slides, Nutrient agar, Mueller Hinton agar, Normal physiological saline, acridine orange/sodium dodecyl sulphate. Media were prepared according to the manufacturer's instruction. Cultural characteristics were observed using standard microbiological techniques. Pure cultures were isolated, Gram stained, followed by biochemical tests to identify the isolates. All glass wares were washed with detergent, rinsed in distilled water and sterilized at 100°C in the hot air oven for 1 hour before use while wire loops were sterilized by passing them through a Bunsen burner flame until it was red hot before use.

## RESULTS AND DISCUSSION

One hundred and nine clinical samples were obtained during the study period and these consisted of 44 urine samples, 20 wound samples, 17 high vagina swabs, 3 urethral swabs, 9 sputum samples, 6 semen samples, 5 stool samples, 2 each of endo-cervical swab and ear swabs and 1 throat swab (see table 1). Overall, the prevalence of *E. coli* organism in the clinical samples was 33.03%. This is higher than the 29% reported for all uropathogens in a study conducted in a tertiary care hospital in Eastern India [16] but lower compared to the 70 - 95 % reported by most scientific reports [27]. Compared to findings in Nigeria, the observed prevalence of *E. coli* is high compared to the 24.5% reported for Specialist Hospital in Yola, Adamawa state [28] but low compared to the 35.5% and 37% reported for tertiary hospitals in Benin, Edo State [29] and Abuja [30] both in Nigeria. The variations between the present study and those within and outside the country may be due to geographical differences and difference in time period during which the studies were conducted. By and large, the prevalent rate observed suggests *E.*

*coli* to be the commonest agent causing urinary infection.

*E. coli* is a Gram negative Enterobacteriaceae. The results of this study showed that *E. coli* was associated with several clinical samples but was most prevalence in stool sample (60.0%) and this was followed by ear swab (50.0%), sputum (44.44%), urine (43.18%), wound (25.0%) and lastly high vagina swab (23.53) (table 1). In line with this observation, Shah et al. (2002) has previously reported *E. coli* to be widely implicated in various clinical infections as hospital acquired and community infections. Indeed, studies have documented *E. coli* as the dominate microbes in many clinical samples and as a major cause of uropathogens by several studies [31, 32]. The observed higher representation of *E. coli* in stool sample in this study further justifies the fact that *E. coli* is an enterobactaiacea.

The antibiogram typing of the isolated *E. coli* (indicated by the Ajumali's coding) showed that no two *E. coli* isolate has same prototype within and between clinical samples (table 2). This has been previously observed by Momoh et al. [29] and was suggested to indicate higher resolving strain differentiation. The variations in *E. coli* prototype with antibiogram analysis is an indication of variation in phenotype between *E. coli* isolated from different individuals and different clinical samples. By implication, the 36 isolated *E. coli* organisms are phenotypically different from one another, even though they are of the same species. This may account for the variations in antibiotic responses reported by different studies, from different geographical location and from different clinical samples.

On antibiotic sensitivity, the findings of this study showed that *E. coli* isolates are highly resistance to many of the available antibiotics (see table 2 and figure 1). In fact, except for Gentamicin (55.56%), Nitrofurantoin (55.56%), Ofloxacin (52.78%), Ciprofloxacin (55.56%), Perflacine (61.11%) and Cefotaxime (69.44%), the other fifteen antibiotics herein investigated were more resistance than susceptible to *E. coli* organism (figure 1). Cefotaxime was the most active antibiotic to *E. coli* with a susceptibility potency of 69. 44% while Amoxicillin was the weakest with susceptibility potency of 11.11% (table 2 and figure 1). The findings in this study are in variant with the findings by Olowe et al. [33] who observed antibiotic sensitivity of *E. coli* isolates in Oshogbo, South-west Nigeria as nalidixic acid (86%), gentamicin (60%), cefuroxin (42.2%), co-trimoxazole (41.5%) and sulphnamide (22.2%).

Panta et al. [34] on the other hand reported *E. coli* isolated from different clinical sample in a tertiary hospital in capital of Nepal to show the following resistivity pattern; Amoxicillin (65.5%), Co-trimoxazole (19.5%), Ciprofloxacin (5.3%), Gentamycin (13.3%), Amikacin (51.3%),



Nitrofurantoin (70.8%), Cefotaxime (38.8%) and Ceftriaxone (51.3%). Alo et al. [35] reported antibiotic sensitivity pattern of *E. coli* bacterial isolates from UTIs patients in Federal Teaching Hospital Abakaliki as follows; Gentamycin (35.5%), Ceftriaxon (35.5%), Ciprofloxacin (42.5%), Ofloxacin (28.5), Erythromycin (17.7%), Tetracycline (13.9%), Chloramphenicol (6.9%), Amoxicillin (7.5%), Nitrofurantoin (97.8%), Perfloxacin (52.7%), Cotrimaxole (21.6%), Flucloxacillin (29.8%), Nalidixic acid (80.0%) and Streptomycin (11.3%). The group 6 (Ofloxacin, Ciproxin and Peflacin) and 3 (Genticin, Tobramycin and Streptomycin) antibiotics were the most susceptible to *E. coli* with percentage group susceptibility potentials of 56.48% and 50.93% respectively. On the other hand, the group 1 (Ampicillin, Amoxicillin and Cloxacillin) and 2 (Flucloxacillin, Amoxycylav and Ampisulbactam) antibiotics were the most resistance antibiotics with percentage group susceptibility potential of 20.37% and 21.30% respectively (see table 2 and figure 1).

Of clinical and pharmacological significance, the *E. coli* isolates from different clinical samples in this study showed higher percentage multidrug resistant (figure 2). Similar high resistance pattern observed in this study had been reported by several studies and was concluded that pathogenic isolates of *E. coli* have relatively high resistance potentials for developing resistance [13, 36]. Although all the clinical samples isolates were multidrug resistance, *E. coli* isolates from sputum, ear swab and urine were more multidrug resistance. The observed higher resistance than susceptibility by many antibiotics in this study may be due to inappropriate used of the available antibiotics. This assertion is based on the fact that bacteria have ability to undergo mutation or acquiring resistance gene when antimicrobial agents

are inappropriately used [37]. This observed high and pattern of multidrug resistance in this study therefore indicates threat to antibiotics treatment in the study area. In addition, the genetic and non genetic transfer of resistance that exists in *E. coli* as observed in this study, indicate that the multi-drug resistant bacteria infections is a problem with within developing and developed countries.

The observed high multidrug resistance and antibiotics sensitivity patterns in this study may be due to the variations in phenotype even though the isolates were of the same species. This may also account for the difference in antibiotic susceptibility potency between this study and other studies conducted within and outside Nigeria. This assertion is based on the fact that coliforms have extensively changed their susceptibility patterns [38]. According to Raghunath *et al.* [38],  $\beta$ -lactam resistance is widespread among Coliform bacteria due to vertical as well as horizontally acquired resistance factors. The observations and findings of this study are in line and call for the need for antibiotics surveillance within and between geographical vicinity.

In conclusion, the results therefore support the fact that the potential of antibiotics in the treatment of infectious diseases is threatened and this may be due to: (1) the changing genetic mutation as no two *E. coli* was observed to have same Antibigram type and (2) emerging and spreading strains of multiple resistance microorganisms- specifically *E. coli*. The findings reinforce the needs for active and continuous antibiotics surveillance in the community level, Local Government level, State level and National level. Furthermore, there is need for antibiogram testing prior to antimicrobials prescription.

**Table 1: Distribution of *Escherichia coli* bacteria isolates from specimen according to clinical samples**

	Specimens										Total
	Urine	Sputum	Semen	Stool	ECS	Throat swab	Ear swab	Wound	HVS	Urethral swab	
Total sample collected	44	9	6	5	2	1	2	20	17	3	109
<i>Escherichia coli</i>	19	4	0	3	0	0	1	5	4	0	36
% in specimen	43.18	44.44	0.0	60.0	0.0	0.0	50.0	25.0	23.53	0.0	33.03

**KEY:** HVS = High vagina, ECS = Endo-cervical swab

**Table 2: Antibigram type indicated by Ajumali's coding for *E. coli* isolated from different clinical samples by Ajumali's coding**

Clinical samples		Conc.	NV	Antibiogram type via by Ajumali's coding																				A%MS	
				Penicillin	Ampicillin	Amoxicillin	Floxapen	Amoxiclav	Ampisulbactam	Genticin	Streptomycin	Tobramycin	Tetracycline	Chloramphenicol	Clotrimoxazole	Nitrofurantoin	Metronidazole	Nalidixic acid	Ofloxacin	Ciprofloxacin	Perflacine	Cephalexin	Cefotaxime	Cefuroxime	
			1	10	2	4	1	2	4	1	2	4	1	2	4	1	2	4	1	2	4	1	2	4	
			1000072, 0060002, 0440002, 0400002, 0441000, 1673165, 2077777, 3077266, 0031072, 0040102, 0000542, 0000070, 4650172, 0130002, 0420131, 0010372, 0272770, 0072070, 2051107																				30.93		
			0000132, 0000040, 2022417, 2013206																				21.43		
			0000100, 0052164, 7657577																				44.90		
			2400162																				30.61		
			1525277, 5404170, 2170046, 0010016, 7671117																				44.08		
			0372171, 0270576, 3131176, 0070147																				45.41		
%GS	%DS	nPS	HS	WS	ES	StS	SpS	US																A%MS	

Key: Conc.= drug concentration in µg (Conc./disk), NV= numerical values, US = urine sample, SpS=Sputum sample, StS=Stool sample, ES= Ear swab, WS=Wound sample, HS=High vagina swab sample, A%MS= average percentage multidrug sensitivity. nPS= number of positive *E. coli* bacterial, %DS = percentage drug sensitivity, %GS=percentage group sensitivity.

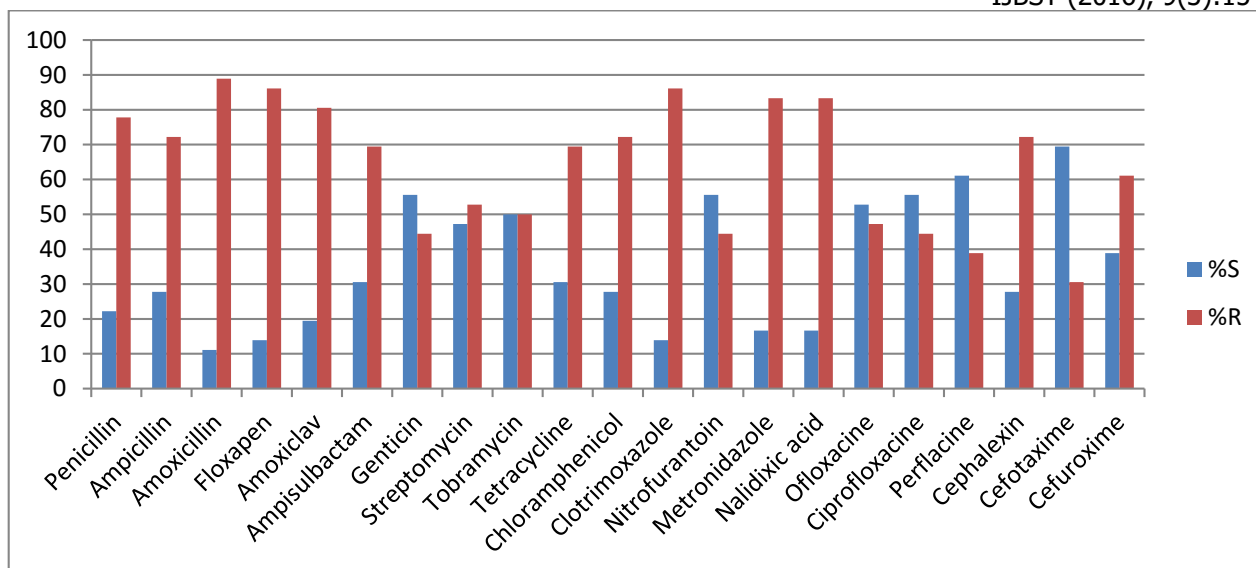


Figure 1. Comparative percentage sensitivity and resistivity of *E. coli* isolates to available antibiotics  
(Key: %S= Percentage sensitivity, %R= percentage resistivity)

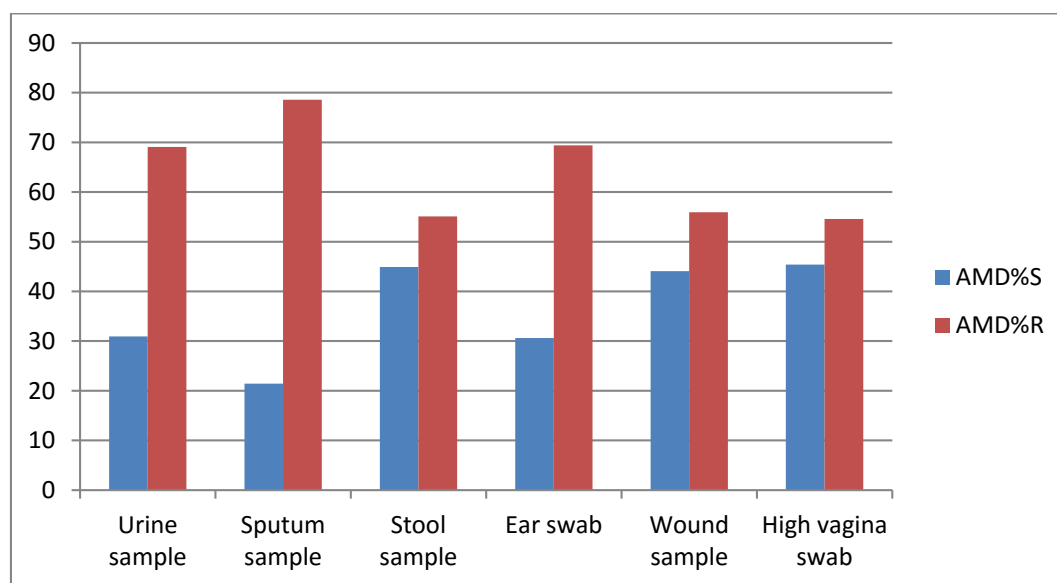


Figure 2. Comparative multidrug percentage sensitivity and resistivity of *E. coli* isolated from different clinical sample  
(Key: AMD%S= Average multidrug percentage sensitivity, AMD%R= Average multidrug percentage resistivity)

### Competing interests

The author declare no competing interest

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